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Preventing the Death of Schwann Cells under High Glucose Conditions is a Promising Approach to Treat Diabetic Peripheral Neuropathy using Chinese Herbal Medicine

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Description

Diabetes Mellitus (DM) is a non-communicable chronic condition. According to a report released in 2021 by the International Diabetes Federation (IDF), diabetes is now a serious health risk for people all over the world and will be one of the global health crises that grow at the fastest rate in the 21st century. Due to its persistent and undetectable onset, diabetic peripheral neuropathy (DPN), one of the significant complications of diabetes, is frequently greatly undertreated. DPN affects more than half of diabetics, and it is linked to a higher risk of lower limb amputation and foot ulceration, as well as significant morbidity and mortality. DPN has emerged as a significant risk factor for disability and death. In addition, the annual increase in the number of DPN patients is seriously jeopardizing the patients' quality of life and putting financial strain on society and the healthcare system [1].

Although controlling blood glucose levels and implementing lifestyle changes are essential to diabetes treatment, many diabetics still develop neuropathy. Patients in the Steno-2 study received multifactorial treatment that included glycemic control, aspirin, statins, renin-angiotensin blockers, and lifestyle modifications; however, they continued to experience a high rate of diabetic complications. Consequently, DPN treatment remains a significant medical challenge despite the fact that numerous studies have been conducted and the findings are extremely instructive. In addition, the complexity of the DPN pathogenesis is unknown to the fullest extent, and there is no universally accepted treatment plan for DPN. As a result, tackling DPN is a major clinical issue and a significant demand that necessitates ongoing, long-term efforts [2].

The majority of glial cells in the peripheral nervous system (PNS) are Schwann cells (SCs). Because the pathological changes that occur in DPN were believed to be primarily axonal degeneration, the majority of clinical and basic research initially focused on the effects on neurons and neglected to investigate the effects on SCs. In patients with DPN, electron microscopy was used to study the peroneal nerve until 1979. Human DPN, exhibits independent changes in axonal degeneration as well as abnormalities in demyelination and SC metabolism. SC developed into a research boom quickly. According to these studies, SCs are an important part of the PNS. Neuronal structure, function, regeneration, and repair following injury are all closely linked to their activation, proliferation, and apoptosis. Axonal degeneration follows segmental axonal demyelination and myelin regeneration as the first pathological process of DPN. Apoptosis in SCs is triggered by hyperglycemia, according to current research. In vivo and in vitro, SCs subjected to hyperglycemia exhibit the typical morphological characteristics of apoptosis. In addition, it is conceivable that SC damage could result in nerve fiber injury, which could be the initial step in the pathogenesis of DPN. As a potential treatment for DPN, the researchers suggested that future research focus on SC recovery and apoptosis inhibition. A novel approach to treating DPN may result from this discovery [3].

Under the direction of TCM theory, traditional Chinese medicines (TCM) are used to prevent, diagnose, treat, or regulate human functions. The core of Traditional Chinese Medicine (TCM) is botanicals, which are comprised of at least 11,146 plant species belonging to 383 families and 2309 genera. CHM has a variety of active ingredients, including extracts and monomers, that can be used to treat diabetes and its complications. As of late, many examinations have exhaustively explored the potential antiapoptotic impacts of CHM on SCs to introduce the restorative impacts unbiasedly and completely. We began this review by describing SCs, including their physiology and function. Then, we talked about how SCs in DPN die off. The various mechanisms by which an extract or monomer derived from CHM inhibits apoptosis were also the subject of discussion. Last but not least, we provided a synopsis of the potential and role of CHM in DPN, which will provide fresh treatment options for DPN [4].

SCs are glial cells that first develop into SC precursors from neural crest cells. After that, they transition into immature SC stages and eventually mature SCs. In the PNS, SCs can be broadly divided into the two categories of mature SCs that follow: SCs with myelination (MSC) and SCs without myelination (NMSC). An NMSC wraps a number of axons (C-fiber) to form a remark bundle, whereas an MSC encases an axon to create a multilayer myelin sheath. Together, the two kinds of SCs cover the axons of peripheral nerves. This provides a shield that protects axons, facilitates nerve impulse conduction, and provides nutritional support [5].

A membrane that wraps around the axon to protect it is called a myelin sheath. SCs develop with axons in the PNS. The SC surface is pressed into a longitudinal groove that houses axons during the growth process. The mesaxon is a membrane that connects the cytosol at the sulcus's edge. After that, the mesaxon lengthens the axon and coils it multiple times, creating myelin sheaths made of many concentric layers of spiral membranous plates. As a result, the SCs' cell membrane forms the PNS's myelin sheath, which is a part of the SCs. The nerve's function depends on the encasement of axons. The most fundamental function of the SC in an intact nerve is to act as an insulating barrier, preventing the movement of ions in the intersegmental area and allowing the rapid transmission of depolarizing currents along the axon and across the membrane only from Ranvier nodes.

Conclusion

This enables saltatory conduction to transmit sensory and motor information quickly and effectively. SCs can impressively emit numerous trophic variables and cell attachment particles that can influence the development of neurons. Numerous known neurotrophic molecules, including ciliary neurotrophic factor (CNTF), brain-derived neurotrophic factor (BDNF), and nerve growth factor (NGF), have been identified as coming from SCs in previous research. The successful co-culture model of SCs and neurons demonstrated that SCs are capable of supplying neurons with trophic factors and fostering their growth. Additionally, the neuron is a structure that requires a lot of energy. The mitochondria of SCs are also involved in the neuron's energy metabolism, as previously discovered in studies of axonal mitochondria. An autopsy of human

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tissue showed that abnormal changes in SC mitochondria accompany axonal lesions in many peripheral nerve diseases. As a result, axons rely heavily on the SCs' metabolic equilibrium.

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Conflict of Interest

None.

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